ABSTRACTS

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RADON EXPOSURE IN AN ONGOING LUNG-CANCER CASE-CONTROL STUDY IN MISSOURI *(Thurs., 11:15 a.m.)*

M. C. R. Alavanja,[1] R. Brownson,[1] J. A. Mahaffey,[2] and J. D. Boice, Jr.[3]

[1]National Cancer Institute, Bethesda, Maryland; [2]Pacific Northwest Laboratory, Richland, Washington; and [3]Missouri Department of Health, Columbia, Missouri

Year-long radon measurements were made in the homes of 400 women participating in an ongoing case-control study of lung cancer in Missouri. Alpha-track detectors were placed in the bedroom and kitchen of every home occupied by the study subjects for the past 30 y (mean, 2.6 homes). The length of residence in the subjects' current home was 17.1 y on average. Overall, the arithmetic and geometric mean concentrations of radon were 1.8 and 1.2 pCi L^{-1} (67 and 44 Bq m^{-3}), respectively. Concentrations greater than 4 pCi L^{-1} (148 Bq m^{-3}), the Environmental Protection Agency's action level, were found in 8.7% of the homes; 1.4% exceeded 8 pCi L^{-1} (296 Bq m^{-3}). However, the proportion of women exceeding a 30-y time-weighted-average concentration of 4 pCi L^{-1} significantly decreased with increased number of residences. For those living in 1, 2, and 3+ homes during the last 30 y, percentages greater than or equal to 4 pCi L^{-1} were 15.4%, 11.4%, and 0%, respectively.

These findings support recent theoretical calculations that point to a substantial reduction in statistical power to detect a radon-related lung-cancer risk in populations with high residential mobility.

A second approach to estimating cumulative radon exposure is also being evaluated. Allyl diglycol carbonate (CR-39) (plastic etched-track) detectors are affixed to glass objects, such as mirrors, that have been in the possession of study subjects for at least 20 y. We will measure alpha-particle emissions from long-lived radon daughters that have accumulated in the surface of these glass objects.

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MEASUREMENTS AND MODELING OF RADON INFILTRATION INTO A TEST DWELLING*

(Wed., First Group, 8:40 a.m.)

F. J. Aldenkamp, P. Stoop, E. T. Loos, R. J. de Meijer, and L. W. Put

Kernfysisch Versneller Instituut, Rijksuniversiteit Groningen, The Netherlands

To understand radon transport in Dutch residences, we made a detailed study of radon characteristics in a test residence. Radon source strengths were determined by *in situ* exhalation measurements. Information on the dynamic aspects of radon transport was obtained from continuously monitoring the radon concentration in various compartments, leakage parameters, temperature, pressure differences between compartments, and the height of the ground water table. Source strengths and dynamic variables were used as input for a multi-compartment model to describe the variations and interrelations of radon concentrations in various compartments.

Depressurization and pressurization of the crawl space led to substantial changes in the radon concentration in the crawl space but had a reduced effect in the living room. The results suggest that pressure-driven flow of radon through the soil is an important factor in radon concentration in the crawl space. However, the transport of radon-rich air, via the cavity wall, appears to be a possible source of radon in the living room.

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*Research supported in part by the Dutch Government and in part by the Commission of the European Communities.

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ANOMALOUS INDOOR RADON CONCENTRATIONS IN A RESEARCH REACTOR BUILDING *(Wed., First Group, 1:50 p.m.)*

M. Balcázar, A. Chávez, and A. López

Instituto Nacional de Investigaciones Nucleares, Mexico, DF, Mexico

Anomalously high indoor radon concentrations have been measured in a research reactor building at the Nuclear Center of Mexico. Measurements were carried out during major maintenance, when some areas were closed, and ventilation was not working properly. Radon and its decay products were detected using a passive, thin, solid-state nuclear track detector whose minimum detection limit for a 1-month exposure time is 10 Bq m^{-3}.

We will discuss radon contributions from building materials, ventilation, and implications for long-term occupational cancer risk evaluation.

DETERMINATION OF RADON-222 IN GROUND WATER USING LIQUID SCINTILLATION COUNTING-SURVEY OF CAREFREE-CAVE CREEK WATER BASIN IN ARIZONA *(Wed., First Group, 1:25 p.m.)*

J. M. Barnett,[1] J. W. McKlveen,[1] and W. K. Hood, III[2]

[1]Arizona State University, Tempe, Arizona; and [2]Arizona Department of Environmental Quality, Phoenix, Arizona

Well water used in homes may contribute additional ^{222}Ra to the indoor radon concentration. Our research objectives are to establish a method to measure radon in ground water using liquid scintillation (LS) spectrometry, and to determine the lung dose from the radon released into the air.

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The method involves collecting a nonaerated, slow, steady flow of water from a pumping well into a 437-ml (16-oz) glass bottle. A high meniscus assures no head space, and the sample is capped. In the laboratory, standard 22-ml LS glass vials are filled with 10 ml of a toluene-based mineral oil LS cocktail and two 5-ml sample aliquots. The vial is capped tightly, shaken vigorously, and placed in the LS counter.

Equilibrium was established in about 3.5 h, after which samples were counted for 100 min each. Only radon and daughters were measured. According to NUREG/CR-4007, the lower limit of detection is 1.9 Bq L^{-1} (51 pCi L^{-1}) in the window of interest. The radon and progeny detection efficiency was between 320 and 330%, and the average background was approximately 6 counts per minute.

We expect that wells containing radon concentrations between 100 and 1000 Bq L^{-1} would produce an effective dose equivalent to the lungs of 0.4 to 0.7 mSv y^{-1} (40-70 mrem y^{-1}). Our study of 28 wells in Carefree-Cave Creek indicates that in 25% of the wells, radon levels were over 100 Bq L^{-1} (2700 pCi L^{-1}). Twelve wells were chosen for monthly monitoring to ensure the efficacy of the methodology. This simple method allows us to count a large number of samples over a short time period.

RADON EXHALATION RATE FROM COAL ASH AND BUILDING MATERIALS IN ITALY *(Poster, Wed., First Group, 2:15 p.m.)*

A. Battaglia,[1] D. Capra,[1] G. Queirazza,[2] and A. Sampaolo[3]

[1]CISE SpA, Segrate, Italy; [2]ENEL/CRTN, Milano, Italy; and [3]ENEL CRC, Brindisi, Italy

The Italian National Electricity Board (ENEL), in cooperation with CISE, initiated an investigation program to assess the hazard

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connected with using coal ash as a partial substitute for concrete and other building materials.

We investigated the natural radioactivity levels of more than 200 building materials. The survey involved materials available in Italy, categorized by geographical location and type of production. We also examined approximately 100 samples of fly ash from U.S. and South African coal, obtained from Italian power plants.

Exhalation rates from about 50 powdered materials were determined by continuously measuring radon concentration growth in closed containers. Measurements were also performed on whole brick, slabs, and tiles. Details about the high-sensitivity measuring devices are presented.

The influence of fly ash on exhalation rates was investigated by accurately measuring radon emanation from slabs with various ash/cement ratios and with slabs of inert materials having various radium concentrations.

We will discuss results of forecasting indoor radon concentrations under different ventilation conditions. Two identical test rooms are being built, one with conventional and one with fly-ash building materials, to compare theoretical calculations and experimental data. Specifications for instruments to control and to measure the most important parameters are also discussed.

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PARAMETERS THAT CHARACTERIZE THE RADON HAZARD OF SOILS* *(Wed., First Group, 9:55 a.m.)*

T. E. Blue, M. S. Jarzemba, and J. A. Mervis

The Ohio State Nuclear Engineering Program, Columbus, Ohio

There is little correlation between radon concentrations in soil and radon concentrations in homes. One explanation is that the soil radon concentration does not fully characterize the soil as a radon hazard. We have identified important soil properties by mathematically modeling ventilated air enclosed in basement walls of thickness T (through which radon convects) and surrounded by soil of infinite extent (through which radon diffuses). The radon instantaneously mixes uniformly with the basement air and is lost from the basement air by ventilation (λ_v) and decay (λ). The steady-state solution for the radon concentration in the basement is:

$$C_1 = \frac{C_s K\Delta P}{(\lambda+\lambda_v)\mu T}\,\frac{A}{V}\left[\epsilon_3 L_3 \Big/ \left(\epsilon_3 L_3 + \frac{K\Delta P}{\mu T\lambda}\right)\right]\exp[-\lambda\mu\epsilon_2 T^2/(K\Delta P)]. \quad (1)$$

In Equation 1, C_s, L_3, and ϵ_3 are the soil's radon concentration, radon diffusion length, and porosity; ϵ_2 and K are the wall's porosity and permeability; μ and ΔP are the wall air's viscosity and pressure difference across the basement wall; and V and A are the basement's volume and wall surface area. From Equation 1, for a tight home (i.e., $K\Delta P/\mu T\lambda << \epsilon_3 L_3$), it is sufficient to simply measure C_s to characterize the radon hazard of a soil. If a home is very leaky, (i.e., $K\Delta P/\mu T\lambda >> \epsilon_3 L_3$), then $C_s\epsilon_3 L_3$ is a more appropriate parameter for assessing the radon hazard.

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*Work supported by the Ohio Air Quality Development Authority.

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CHARACTERIZATION OF RADON ENTRY RATES AND INDOOR CONCENTRATIONS IN UNDERGROUND STRUCTURES* *(Wed., First Group, 11:30 a.m.)*

T. B. Borak, F. W. Whicker, L. Fraley, M. S. Gadd, S. A. Ibrahim, F. A. Monette, R. Morris, and D. C. Ward

Colorado State University, Ft. Collins, Colorado

An experimental facility has been designed to comprehensively determine the influence of soil and meteorological conditions on the transport of radon into underground structures. Two identical basements are equipped to continuously monitor pressure differentials, temperatures, soil moisture, precipitation, barometric pressure, wind speed, wind direction, natural ventilation rates, and radon concentrations. A computerized data acquisition system accumulates and processes data at the rate of 6000 points per day. The experimental design is based on performing experiments in one structure, with the other used as a control. Indoor radon concentrations have temporal variations ranging from 150 to 1400 Bq m^{-3}. The corresponding entry rate of radon ranges from 300 to 10,000 Bq h^{-1}. It is interesting that when the radon entry rate is high, the indoor radon concentration decreases, whereas elevated radon concentrations seem to be associated with slow but persistent radon entry rates. This inverse relationship is partially due to compensation from enhanced natural ventilation during periods when the radon entry rate is high. Correlations between measured variables in the soil and indoor-outdoor atmospheres are used to interpret these data. This laboratory has the capability to generate essential data required for developing and testing radon transport models.

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EVALUATING CHROMOSOME DAMAGE TO ESTIMATE DOSE TO TRACHEAL EPITHELIAL CELLS*

(Wed., Second Group, 10:25 a.m.)

A. L. Brooks,[1] K. Rithidech,[2] N. F. Johnson,[3] D. G. Thomassen,[3] and G. J. Newton[3]

[1]Pacific Northwest Laboratory, Richland, Washington; [2]Brookhaven National Laboratory, Upton, New York; and [3]Lovelace, Inhalation Toxicology Research Institute, Albuquerque, New Mexico

Chromosome aberration frequency has been widely used to estimate dose in radiation accidents when no physical dosimetry is available. To use cells for this purpose, it is essential to understand their radiation sensitivity. Basal and secretory cells are thought to represent the major cells at risk in the upper respiratory tract for the induction of cancer. Using an *in vivo/in vitro* approach, we have evaluated cell killing and chromosome aberrations to determine radiation sensitivity and to estimate the dose to epithelial cells following radon inhalation.

Tracheal epithelial cells were isolated, sorted by flow cytometry into populations of basal and secretory cells, grown in serum-free medium, and exposed to graded doses of x rays. Cell survival and chromosome aberration frequency were determined in both types of cells. No differences in the survival response were observed for the two cell types. A 2-Gy dose of x rays induced 0.52 ± 0.07 or 0.56 ± 0.07 aberrations/cell in the basal and secretory cells, respectively. These data suggest that the radiation sensitivity is similar for the two cell populations and that total aberration frequencies can provide an estimate of radiation dose.

In other experiments, two groups of F-334/N rats inhaled radon and its progeny at concentrations and times to produce total exposures

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of either 900 or 1000 working level months (WLM). Rats in the first group were sacrificed at 4 h after the end of radon inhalation; those in the second group were allowed to recover for 8 or 32 days after inhalation. Control rats were sacrificed on the same schedule.

At necropsy, tracheal epithelial cells were isolated and grown in serum-free medium; chromosome damage was measured after *in situ* fixation. Aberration types, their distribution in the cell population, and frequency were determined at each sacrifice time.

At day 0 after the 900-WLM exposure, the frequency was 0.94 aberrations/cell, with a distribution index, μ, of 8.1. This frequency indicates that chromosome damage was heavy and that, relative to the Poisson distribution, initial damage was highly dispersed in the cell population.

At 8 and 33 days after exposure to 1000 WLM, the aberration frequency was 1.3 and 0.62, and the distribution index was 9.5 and 8.7, respectively. The distribution was, again, nonrandom, and aberrations were retained in the cell population with a long effective half-life.

These data demonstrate that, in future studies, doses of much less than 1000 WLM and low dose rates can be used to study radon-induced damage and, furthermore, that aberrations provide a useful index of damage. By comparing the aberration frequency in epithelial cells with dose-response relationships observed for cells exposed *in vivo* and *in vitro* to alpha particles, it was possible to estimate the average dose to tracheal epithelial cells from radon and radon progeny inhalation. From these calculations we estimated that tracheal cells received 0.6 to 1.6 Gy following inhalation exposure to 900 WLM.

Cellular damage can help validate the distribution of dose in respiratory epithelium, can provide information on exposure-dose relationships, and furnish additional data on radon risk estimates.

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CONTROL OF OCCUPATIONAL EXPOSURE TO RADON IN THE WORKPLACE *(Tues., 8:45 a.m.)*

L. D. Brown

Saskatchewan Human Resources, Labor and Employment, Regina, Saskatchewan, Canada

Mining high-grade uranium ore is an important industry in Saskatchewan; inhalation of radon progeny is therefore a major concern. Strenuous efforts by the Provincial Radiation Safety Unit have kept the exposures received by the great majority of miners to less than one working level month (WLM) in a year. Many workers in non-radiation-related employment may be receiving comparable, or occasionally higher, exposures from radon levels in the buildings where they work. Because this cannot easily be justified, a draft regulation has been introduced to limit to 1 WLM the annual radon-progeny exposure received by any worker as a result of high natural radon levels in his place of employment.

The Occupational Health Branch is anxious to assess the potential impact of this proposed regulation before it is formally adopted by measuring radon levels in a representative group of buildings selected at random from all parts of the province. Schools have been chosen for this purpose because they provide a convenient sample size, are architecturally very similar to many typical work-places, represent buildings of all ages, are uniformly distributed in all habitable areas, and, in any given community, have a fairly constant numerical relationship to the number of residential buildings. Initially, single readings are being made in each school, using the charcoal canister procedure. Wherever a nontrivial initial reading is obtained, follow-up samples are taken as necessary. This paper reviews the progress of the study and reports the measure-ments so far completed.

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ALPHA PARTICLE SPECTRA AND MICRODOSIMETRY OF RADON DAUGHTERS *(Tues., 9:10 p.m.)*

R. S. Caswell and J. J. Coyne

National Institute of Standards and Technology, Gaithersburg, Maryland

We are interested in understanding the physics of the process by which radon-daughter alpha particles irradiate cells leading to the induction of cancer. We are focusing initially on two aspects: the alpha spectra incident upon cells, which are needed for input to biophysical models of cancer induction; and microdosimetric spectra and parameters which give information on radiation, quality. Adapting an analytical method previously developed for neutron radiation, we have calculated the alpha-particle slowing-down spectra (the spectra incident upon cells) and, subsequently, the microdosimetric spectra and parameters for various cell nucleus or site diameters. Results will be presented from three "MODEs" of program operation. MODE 1 is for the thin, plane source of radon-daughter activity adjacent to the epithelium. MODE 2 is for the thick source layer (the mucus-serous layer) adjacent to the epithelium. MODE 4 is for cylindrical airways of various radii lined by the mucous-serous layer. MODE 1 is most useful for developing an understanding of the problem, while MODE 4 is most anatomically relevant. MODE 3 is not discussed in this paper.

Alpha-particle spectra and microdosimetric spectra and parameters are studied as a function of cell depth, $^{218}Po/^{214}Po$ ratio, airway radius, and cell nucleus or site size. Mean dose as a function of depth below the airway surface is also available from the calculation. We make these results available through a published compilation and personal computer diskettes. We are commencing comparison studies with calculations of other workers, where available, and plan to extend the calculations to the nanometer-target level.

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RADON UPTAKE IN HUMANS *(Tues., 8:10 p.m.)*

G. R. Chakravarthy,[1] G. I. Lykken,[2] and J. J. Worman[2]

[1]Radiation Oncology, United Hospital, Grand Forks, North Dakota and [2]University of North Dakota, Grand Forks, North Dakota

As early as 1967, it was reported that the equilibrium concentration of radon in the human body is about 30% of that in the air, and concentrations in fatty tissue may be 700%. Furthermore, about one-third of the inhaled radon decay products pass from the lungs into the bloodstream to irradiate, in particular, the brain, bone marrow, nervous system, and lipoids (lipids). Most researchers, however, ignore these facts and maintain that radon gas flows quickly in and out of the lungs, never lingering long enough in the body to cause damage, while the radon progeny, attached to particulate matter, lodge in the bronchial tree, where their emissions may produce lung cancer.

Recent studies on cancer incidence as related to radon in potable water indicate that radon released from the water and breathed into the lungs may be responsible for certain cancers. Therefore, we performed experiments to study radon uptake into the body through the lungs.

Two human subjects were exposed to radon inhalation of a known concentration. Two sets of data were obtained: (a) after each of several radon-laden breaths, the exhaled air was collected, and (b) washout of radon in successive breaths was measured after a single radon-laden breath. From the results we conclude that: (1) Radon is dynamically taken up by the lungs; (2) radon is not washed out, as commonly believed. Further studies are in progress using a laboratory human lung model.

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EFFECTS OF EXPOSURE UNCERTAINTY ON ESTIMATION OF RADON RISKS *(Thurs., 3:45 p.m.)*

D. B. Chambers,[1] L. M. Lowe,[1] P. M. Reilly,[2] and P. Duport[3]

[1]SENES Consultants, Ltd., Richmond Hill, Ontario, Canada; [2]University of Waterloo, Waterloo, Ontario, Canada; and [3]Atomic Energy Control Board, Ottawa, Ontario, Canada

Estimates of lung-cancer risk from exposure to radon daughters are largely based on epidemiological studies of underground miners. The reliability of exposure data for these miners is a cause for concern, as actual workplace measurements of radon and/or radon-daughter levels are either sparse or absent for the early years of mining, when much of the exposure occurred.

This paper reviews the factors which affect exposure estimation, including the temporal and spatial variability of radon and radon-daughter levels in the workplace; changes in mining methods and ventilation practices; and uncertainties about the miners' work histories. Available data for the following study groups are reviewed in detail: Colorado (USA) uranium miners; Ontario, Canada, miners (uranium, with and without gold-mining experience); Czechoslovakian uranium miners; Eldorado uranium miners (Port Radium and Beaverlodge, Canada); Newfoundland, Canada, fluorspar miners; and Malmberget, Sweden, iron miners. Our review suggests that data for the Colorado miners, the Ontario miners, and the Czechoslovakian miners currently provide the best basis for risk estimation.

We present two statistical approaches, both based on Bayesian methods, which permit evaluation of the effect of uncertainty in exposure (measured in working level months [WLM]) on the risk coefficients). The first method requires only an estimate of the cumulative WLM exposure of a group of miners, an estimate of the number of excess lung cancers, and a specification of the uncertainty about the cumulative exposure. This method assumes a linear

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relationship between increasing WLM exposure and risk, and provides an estimate of the posterior probability density function of the risk coefficient (with units of excess cases per WLM).

The second technique is based on a linear regression model which incorporates errors (uncertainty) in the independent variable (WLM) and allows the dependent variable (cases) to be Poisson distributed. This method permits calculation of marginal posterior probability distributions for either the slope (i.e., the risk coefficient) or the intercept. The regression model has been applied to the data sets noted above.

None of the analyses performed on the data sets is inconsistent with a linear exposure-effect relationship. Based on analyses of these miner groups, the most likely ranges for the absolute and relative risk coefficients are from <5 to about 15 cases per 10^6 person-y per WLM, and from 0.5 to about 1.5% per WLM, respectively.

MUTAGENIC EFFECTS OF ALPHA PARTICLES IN NORMAL HUMAN FIBROBLASTS* *(Wed., Second Group, 9:15 a.m.)*

D. J. Chen, T. Hanks, S. Carpenter, and K. Tsuboi

Los Alamos National Laboratory, Los Alamos, New Mexico

Alpha-irradiation of the bronchial airways from inhaled radon progeny increases the risk of developing lung cancer, although molecular mechanism of radon-induced lung cancer is not clear at this time. However, one of the most important genetic effects of ionizing radiation is the induction of mutation. Therefore, our objective is to use a well-defined model system to determine the mutagenic potential of alpha-particles in normal human skin cells at both the cellular and molecular level.

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Normal human skin fibroblasts were irradiated with alpha particles (3.7 MeV, LET 100 keV/μm) from ^{238}Pu. Mutagenicity was determined at the X-linked hypoxanthine-guanine phosphoribosyl transferase (HPRT) locus.

Our results indicate that alpha particles are more efficient in mutation induction than gamma rays. Our results on repair of mutational damage suggest that there is a considerable decrease in mutation induction by gamma rays following postirradiation repair. However, there is no evidence for mutational damage repair after alpha-particle exposure.

HPRT-deficient mutants, which are resistant to 6-thioguanine, were isolated and analyzed by the Southern blot technique. Using this technique, we have characterized 50 gamma-ray-induced and 160 alpha-particle-induced HPRT-deficient mutants. Our data indicate that more than 50% of all gamma-ray-induced mutants have a normally structured HPRT gene, and the remaining mutants (45%) contain either a rearrangement, partial deletion, or total deletion of the HPRT gene. In contrast, about 50% of alpha-particle-induced human HPRT mutants contain a total deletion and only 30% contain a normal Southern pattern of the HPRT gene. Our results support the theory that high-LET radiations produce more unrepaired or misrepaired DNA damage than do gamma rays.

PLATE-OUT RATES OF RADON PROGENY AND PARTICLES IN CHAMBERS* *(Tues., 10:40 a.m.)*

Y. S. Cheng and Y. F. Su

Inhalation Toxicology Research Institute, Albuquerque, New Mexico

In indoor and mining environments, deposition or "plate-out" of radon progeny on walls occurs simultaneously with attachment to airborne

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particles. Both airborne particles and walls provide large surface areas on which the highly mobile species can deposit. In addition, the airborne particles will also plate-out, although to a lesser degree, depending on the particle size. The measured plate-out rates for radon progeny have a wide range of values that reflect the room or chamber conditions. Deposition models show that turbulence and natural convection in a room are major factors influencing the plate-out rates. We report plate-out measurements for radon progeny and ultrafine particles in chambers under controlled laboratory conditions. We used a 162-L spherical chamber and monitored the temperature and velocity profile near the chamber wall. Two chamber conditions, with and without a mixing fan (to create turbulence), were studied. Radon-220 progeny were generated from a dry source, and monodisperse particles ranging from 4 to 1000 nm were produced by an electrostatic classifier. A combination of a 50-mesh screen and a filter was used to determine both particle size and concentration. The aerosol rate constants were derived and compared with the turbulent deposition model proposed by Crump and Seifeld. Nonlinear regression was performed to obtain the best values for eddy diffusivity, which is generally expressed as $K_e x^n$, where K_e is the turbulent intensity parameter, and x is the distance from the wall. Summarized values reported in the literature for n and K_e obtained under different conditions were analyzed. The information obtained should improve our understanding of the plate-out process and will be used to predict the behavior of radon exposure atmospheres in indoor environments.

EXPANDED TESTS OF THE LINEAR/NO-THRESHOLD THEORY OF RADIATION CARCINOGENESIS

(Thurs., 2:35 p.m.)

B. Cohen

University of Pittsburgh, Pittsburgh, Pennsylvania

In 1989 we reported on the correlation between male and female age-adjusted lung-cancer rates in 411 U.S. counties, based on

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39,000 measurements, using multiple regression analysis with nine potential socioeconomic confounding factors. Our measurement data base has now been expanded to 175,000 measurements, giving data on 965 counties (913 if we exclude Arizona, California, and Florida), and the multiple regression analysis has been expanded to include 17 confounding factors. Data are fitted to

$$m = a + br + C_1F_1 + C_2F_2 + \ldots + C_{17}F_{17},$$

where m is the lung cancer rate, r is mean radon level, F_i are confounding factors, and a, b, $C_1 \ldots C_{17}$ are 19 constants estimated from the data.

The results for male lung cancer rates (female rates in parentheses) are that b is negative by 4 (5) standard deviations (SD) and discrepant with the positive slope predicted by BEIR-IV by 16 (14) SD. When data are stratified into quintiles on each of the F_i in turn, 94% of the 170 multiple regressions (17 Fs x 5 strata x 2 sexes = 170) give negative estimates of b, and the largest positive b is less than 30% of the BEIR-IV prediction.

Stratification by geographic regions reduces the negative b to zero–there is a strong negative correlation between lung-cancer rates and radon levels for various regions of the United States, and possible explanations for this will be discussed. However, data for individual regions or even for individual states do not give the positive b predicted by BEIR-IV. Stratification of data for individual regions on each F in turn does nothing to reduce the discrepancy.

Data are also analyzed by states (instead of by counties), which has the advantage that cigarette-sales data are available. Values of b are negative and twice as large as in the analysis by counties, and including cigarette sales in the regressions does nothing to reduce the large discrepancy with BEIR-IV.

Data from Environmental Protection Agency surveys, now available for 22 states, are analyzed similarly, and the results of multiple regressions are in good agreement with those from our data for those states.

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The validity of this type of ecological study will be compared with case-control studies as a test of the linear/no-threshold theory.

VALIDITY OF RESIDENTIAL RADON EPIDEMIOLOGICAL STUDIES: A FUNCTION OF STUDY DESIGN

(Thurs., 3:20 p.m.)

S. M. Conrath

U.S. Environmental Protection Agency, Washington, DC

The basic epidemiological study design types will be reviewed. The advantages and disadvantages of their application to the study of residential radon will be discussed.

Particular emphasis will be placed on the problems inherent in incomplete study designs such as the ecological studies and proportional studies which have been widely quoted in the media.

EXPERIMENTAL ANIMAL STUDIES OF RADON AND CIGARETTE SMOKE*

(Thurs., 9:40 a.m.)

F. T. Cross, G. E. Dagle, R. A. Gies, L. G. Smith, and R. L. Buschbom

Pacific Northwest Laboratory, Richland, Washington

Cigarette-smoking is a dominant cause of lung cancer and confounds risk assessment of exposure to radon decay products. Evidence in humans on the interaction between cigarette-smoking and exposure to radon decay products is limited but suggests that there is synergy. Experimental animal data, in addition to showing

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synergy, also show a decrease or no change in risk with added cigarette-smoke exposures. This paper reviews previous animal data developed at Compagnie Générale Des Matières Nucléaires (COGEMA) and Pacific Northwest Laboratory (PNL) on mixed exposures to radon and cigarette smoke, and highlights new initiation-promotion-initiation (IPI) studies at PNL that were designed within the framework of the two-mutation carcinogenesis model. We will present the PNL exposure system, experimental protocols, dosimetry, and biological data observed to date in IPI animals.

MORPHOLOGY OF RESPIRATORY TRACT LESIONS IN RATS EXPOSED TO RADON/RADON PROGENY*

(Wed., Second Group, 1:55 p.m.)

G. E. Dagle, F. T. Cross, and R. A. Gies

Pacific Northwest Laboratory, Richland, Washington

We will discuss the morphologic features of the lesions in the respiratory tract of rats exposed to radon and radon progeny. Groups of male Wistar rats were exposed to 10 to 1000 working levels (WL) of radon progeny in the presence of less than 1 to about 15 mg/m^3 uranium ore dust. Cumulative exposures ranged from 20 to approximately 10,000 working-level-months (WLM). Higher exposure levels produced radiation pneumonitis characterized by interstitial fibrosis, associated with alveolar epithelial cell hyperplasia and accumulations of alveolar macrophages containing phagocytosed uranium ore dust. Nodular fibrosis and alveolar proteinosis were correlated with depositions of uranium ore dust. Vesicular emphysema also occurred at higher exposure levels. Pulmonary adenomatosis appeared to be a preneoplastic lesion; it was composed of nodular proliferation of bronchioloalveolar epithelium without disruption of the general architecture of the parenchyma.

*Work supported by the U.S. Department of Energy under Contract DE-AC06-76RLO 1830.

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At lower levels, rats lived longer than 1 y, and lung tumors and a few tumors of the nasal cavity developed. The principal lung tumors were pulmonary adenomas, bronchioloalveolar carcinomas, papillary adenocarcinomas, epidermoid carcinomas, and adenosquamous carcinomas. Occasionally, malignant mesotheliomas and sarcomas were also present. The malignant lung tumors were characterized by invasion and occasionally metastasized to regional lymph nodes. The relationships of radiation exposure with incidence and types of lung tumors establish the validity of this animal model for studying pulmonary carcinogenic risk in humans.

THE EFFECT OF HOME WEATHERIZATION ON INDOOR RADON CONCENTRATION *(Tues., 9:35 a.m.)*

G. W. Egert,[1] R. L. Kathren,[2] and F. T. Cross[3]

[1]Westinghouse Hanford Company, Richland, Washington; [2]University of Washington, Seattle, Washington; and [3]Pacific Northwest Laboratory, Richland, Washington

In an effort to reduce energy consumption costs, increasing numbers of homeowners are weatherizing their homes to minimize the loss of heated and cooled air to the outside. These house-tightening measures decrease the natural infiltration rate of fresh air into the house, potentially increasing the concentration of indoor pollutants, including the radioactive gas, radon.

We measured radon concentrations with track-etch detectors in 17 wooden frame homes for a period of 3 months before and after weatherization. An additional 42 homes that were not weatherized were also sampled; they constituted the control group. The measured concentrations in the weatherized homes, the control group, and in both groups combined were described by log-normal distributions. The differences between final and initial concentrations approximate both a log-normal and a normal distribution. Student's *t*-test and the Wilcoxon Rank Sum Test of both log-normal and

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normal-data distributions at the 0.05 significance level show an increase in the indoor radon concentration following home weatherization of 40% and 60%, respectively. This suggests that standard weatherization techniques may increase indoor radon levels by approximately 50%.

AN EXPERIMENTAL FACILITY TO SIMULATE RADON-PROGENY BEHAVIOR IN DWELLINGS *(Tues., 11:05 a.m.)*

P. Eklund and M. Bohgard

Lund Institute of Technology, Lund, Sweden

We have built a room (volume, 20 m^3) to simulate domestic exposure conditions for studying the interaction between radon daughters and ambient aerosols. The room has its own ventilation system, in which the air exchange rate can be adjusted between 0 and 5 air changes h^{-1} (ach). The air supply can accommodate low particle concentrations (less than 1 particle cm^{-3} for ventilation rates less than 1 ach). Aerosols can be introduced at any specified particle concentration from 1 to 10^5 cm^{-3}.

Experiments so far have been focused on (1) measuring size distribution of radioactive aerosols by using a differential mobility analyzer combined with track-etch film; (2) studies of the unattached fraction of radon daughters and the impact of the aerosol and induced motion on this fraction, as measured with the single-screen/filter technique; (3) plate-out under various conditions, such as different air flow rates, various aerosol size-distributions and concentrations, and ranges of temperature and humidity, etc. Plate-out is measured using a large-area flow-through pulse ionization chamber. Using this facility, plate-out can be measured even at high particle concentrations.

This large-scale experimental facility is useful for studying the aerosol parameters relevant to domestic radon-progeny exposure.

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RELATIONSHIP OF THE CELLULAR AND MOLECULAR EFFECTS OF ALPHA-PARTICLE IRRADIATION TO RADON-INDUCED LUNG CANCER

(Invited Overview, Wed., Second Group, 8:15 a.m.)

H. H. Evans

Case Western Reserve University, Cleveland, Ohio

The exact processes involved in radon-induced lung cancer have not as yet been delineated, but it is thought that DNA damage induced by alpha particles emitted by radon decay products is responsible for the carcinogenic effects. Although the molecular and cellular damage induced by alpha particles has been found to be similar to those induced by low-LET radiation, the RBE for the alpha-induced effects are greater than 1. The efficiency with which alpha-irradiation induces damage is thought to result from the production of lesions which are irreparable, as evidenced by the deficiency in cellular recovery processes and the presence of a relatively large fraction of unrepaired chromosome breaks and DNA double-strand breaks following exposure of cells to alpha radiation. The irreparable DNA lesions may consist of locally multiply damaged sites caused by the deposition of large quantities of energy in a small volume of DNA. However, even though a fraction of the lesions is irreparable, not all lesions are lethal, since alpha radiation has been found to be both mutagenic and carcinogenic. In some strains of cells, the ratio of mutagenic to lethal lesions is greater after exposure to alpha-radiation than to low-LET radiation. Generally, large deletions have been found in a greater percentage of alpha-radiation-induced mutants than in low-LET radiation-induced mutants. It is possible that the carcinogenic effects of alpha-irradiation are due to deletions which cause the activation of oncogenes or the inactivation of tumor suppressor genes.

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MICRODOSIMETRY OF RADON PROGENY: APPLICATION TO RISK ASSESSMENT* *(Tues., 9:50 p.m.)*

D. R. Fisher,[1] T. E. Hui,[1] V. P. Bond,[2] and A. C. James[1]

[1]Pacific Northwest Laboratory, Richland, Washington; and [2]Brookhaven National Laboratory, Upton, Long Island, New York

We developed methods for calculating radiation doses to individual cells and cell nuclei of human bronchial epithelium from radon and progeny for specified levels of exposure, breathing rates, equilibrium factors, unattached fraction of progeny, and other factors that are important in radon dosimetry. If we also know which cells are likely precursors for cancer and their locations in the respiratory tract, we may then calculate the statistical probability that such cells are irradiated by alpha particles, the number of single alpha-particle hits, and the spectrum of doses delivered (as a probability density in specific energy).

As we continue to study the relationship between microdosimetry and biological effects, we hypothesize that the corresponding probability of lung cancer is related to the specific energy imparted to nuclei of single cells of bronchial epithelium by radon and its progeny. The mathematical relationship could be determined experimentally from irradiations of cultured bronchial epithelial cells and exposures of laboratory animals to radon environments. The fraction of surviving and transformed cells could be compared to the probability density in specific energy for well-defined irradiation conditions by applying the "hit-size effectiveness function" proposed by Bond and Varma, or by unfolding the cell-specific transformation probability from a series of *in vitro* cell-irradiation experiments.

Factors that remain to be quantified are the time course of irradiation, dose rate, linearity of the response at low levels of exposure,

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and relative impact of cofactors (initiators and promoters) which, in addition to the radiation dose, are important in cancer induction.

INVOLVEMENT OF ONCOGENES IN RADON-INDUCED LUNG TUMORS IN THE RAT* *(Wed., Second Group, 1:30 p.m.)*

M. E. Foreman, L. S. McCoy, and M. E. Frazier

Pacific Northwest Laboratory, Richland, Washington

Several oncogenes, notably those of the *ras* and *myc* family, have been implicated in the induction of lung tumors. Although inhalation of radon and radon daughters has been shown to result in a high incidence of lung tumors, the role of oncogenes in these tumors (if any) remains unknown. In certain cases of chemically induced carcinogenesis, unique point mutations in the 12th, 59th, and 61st codons of H-*ras* and Ki-*ras* have been found to transform *ras* photo-oncogenes to dominant-acting oncogenes.

We have isolated DNA from fixed, archived, radon-induced tumors in rats, amplified the oncogene of interest by PCR, and analyzed it by sequencing. Although we have not found any of the classically described point mutations in the H-*ras* gene, preliminary evidence indicates that several common mutations occur with high frequency in the second exon. These point mutations have not been seen in any "spontaneously" occurring tumors. At present we theorize that these mutations represent one of the secondary effects of a multi-step process in the development of these lung tumors. As this project expands, we are making a systematic effort to correlate the molecular data with the pathological data derived from the original studies of these archived tumors.

*Work supported by the U.S. Department of Energy under Contract DE-AC06-76RLO 1830.

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MODELS FOR COMPARING LUNG-CANCER RISKS IN RADON- AND PLUTONIUM-EXPOSED EXPERIMENTAL ANIMALS *(Thurs., 8:50 a.m.)*

E. S. Gilbert, F. T. Cross, C. L. Sanders, and G. E. Dagle

Pacific Northwest Laboratory, Richland, Washington

Epidemiologic studies of radon-exposed underground miners have provided the primary basis for estimating human lung-cancer risks resulting from radon exposure. These studies are sometimes used to estimate lung-cancer risks resulting from exposure to other alpha-emitters as well. The latter use, often referred to as the dosimetric approach, is based on the assumption that a specified dose to the lung produces the same lung-tumor risk regardless of the substance producing the dose.

At Pacific Northwest Laboratory, experiments have been conducted in which laboratory rodents have been given inhalation exposures to radon and to plutonium ($^{239}PuO_2$). These experiments offer a unique opportunity to compare risks, and thus to investigate the validity of the dosimetric approach. This comparison is made most effectively by modeling the age-specific risk as a function of dose in a way that is comparable to analyses of human data. Such modeling requires assumptions about whether tumors are the cause of death or whether they are found incidental to death from other causes.

Results based on the assumption that tumors are fatal indicate that the radon and plutonium dose-response curves differ, with a linear function providing a good description of the radon data, and a pure quadratic function providing a good description of the plutonium data. However, results based on the assumption that tumors are incidental to death indicate that the dose-response curves for the two exposures are very similar, and thus support the dosimetric approach.

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UNCERTAINTIES IN POPULATION RISK ESTIMATES ARISING FROM DIFFERENT CONDITIONS OF EXPOSURE TO INDOOR RADON *(Thurs., 4:10 p.m.)*

R. L. Goble,[1] D. Hattis,[1] and R. Socolow[2]

[1]Clark University, Worcester, Massachusetts; and [2]Princeton University, Princeton, New Jersey

Almost everyone agrees that a risk estimate should be accompanied by a statement about uncertainties. Indeed, for policy purposes, such as setting priorities for actions or for research, the nature and magnitude of the uncertainty may be as important or more important than the risk estimate itself. However, there is surprisingly little agreement about how uncertainties should be analyzed and communicated. Particularly needed are quantitative approaches to uncertainty analysis which systematically combine uncertainties from a number of sources. We illustrate here a general approach applied to a specific group of uncertainties: those resulting from differences in conditions of exposure and in time patterns of exposure, and how they interact within models for estimating indoor radon risk from studies of miners. The approach is based on Monte Carlo simulation over possible exposure conditions and model parameters to obtain distributions of risk estimates. A context for the analysis is provided by an informal survey of uncertainties, from numerous sources, as treated by different research and review groups.

We illustrate results of three types. (1) We show quantitatively how incremental improvements in characterizing exposures will narrow uncertainties in risk estimates for particular population groups. Such improvements may come from better measurements of unattached fractions or from the use of alternative indicators, such as absorbed ^{210}Pb. (2) We characterize the biases in risk estimates which arise from uncertainties in exposure estimates. (3) We describe differences between the risk and uncertainty estimates appropriate for different population groups. In particular, we note that the uncertainties in risk for people having high indoor radon exposures are quite different from those for people with more average indoor exposures.

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ELEVATED INDOOR RADON LEVELS AND ELEVATED INCIDENCE OF LUNG CANCER IN COLUMBUS, OHIO, AND IN FRANKLIN COUNTY, OHIO: CAUSE OR COINCIDENCE? *(Thurs., 10:50 a.m.)*

H. E. Grafton[1] and D. R. West[2]

[1]Indoor Air Consultations, Columbus, Ohio; and [2]Insul-Tech, Inc., Westerville, Ohio

Columbus, Ohio, and Franklin County, Ohio, have been identified as having elevated residential radon levels. Columbus Health Department, Ohio Department of Health, and U.S. Environmental Protection Agency research have shown that average screening measurements for homes in the county range from 63% to 73% above 148 Bq m^{-3}, 23% to 27% above 370 Bq m^{-3}, and 1% above 1850 Bq m^{-3}. For both males and females in the city of Columbus, the observed cancer rate per 100,000 persons for the period 1979-1986 for the bronchi, lungs, and trachea was 62.8; for the state of Ohio, the comparable rate was 49.3. The reliability of residential radon data, the effect of smoking, mobility of residents, and other confounding factors are discussed. We conclude that sufficient evidence does not currently exist to demonstrate a causal or coincidental relationship between elevated radon levels and increased rate of lung cancer based on this type of aggregate data; however, we suggest that Franklin County, Ohio, is an appropriate site for such research.

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RADON: A CAUSATIVE FACTOR IN THE INDUCTION OF MYELOID LEUKEMIA AND OTHER CANCERS IN ADULTS AND CHILDREN? *(Invited paper - Thurs., 2:10 p.m.)*

D. L. Henshaw, J. P. Eatough, and R. B. Richardson

University of Bristol, Bristol, United Kingdom

We have recently performed detailed calculations of the radiation dose to bone marrow from domestic exposure to radon and thoron and their short-lived daughter nuclei. We have taken into account previously unconsidered factors: (1) red bone marrow contains fat cells in varying proportions, depending on skeletal location and age, and in which radon is 16 times more soluble than in the surrounding marrow. The diameter of these cells is typically 100 to 150 μm. Consequently, alpha-particle decays from radon and its short-lived daughter nuclei ^{214}Po and ^{218}Po within the fat cell deliver some of their energy to surrounding marrow and hence to the hemopoietic cells. (2) Room air contains not only pure radon but also radon daughters which may enter the bloodstream, leading to an additional daughter contribution to the marrow dose, as evidenced by observations of enhanced radioactivity in the blood of health spa workers. For thoron, the main dose contribution is from the alpha-decay of ^{212}Po as a result of the presence of the 10.4-h half-life ^{212}Pb in marrow. There is an additional ^{210}Pb dose contribution from ingrowth of ^{210}Pb in bone. The dose values vary strongly with age.

Where radon concentrations are high, these doses to marrow (typically, 1000 μSv y^{-1}) are significant compared to that from low-LET sources. Total low-LET radiation exposure shows relatively little geographical variation, whereas mean international domestic radon concentrations vary by a factor of, say, 10. Therefore, if natural radiation were a major causative factor of cancers, then not only would we have an *a priori* reason for supposing that radon might be associated with the incidence of leukemia and other cancers, but this would be the most sensible variable with which to seek a correlation.

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We have therefore examined international cancer data from up to 15 countries where useful measurements have been published and compared values of population-weighted radon exposure with cancer incidence. Results show that international incidence of myeloid leukemia, cancer of the kidney, melanoma, and certain childhood cancers all show significant correlation with radon exposure in the home.

If these correlations are indicative of cause and effect for myeloid leukemia at all ages, analysis suggests that, overall, 6 to 12% of incidence in the UK may be attributed to radon. In Cornwall, UK, where radon levels are higher than the UK average, the range is 23 to 43%. For the world average radon exposure of 50 Bq m^{-3}, we estimate that 13 to 25% of myeloid leukemia at all ages could be caused by radon. We also estimate that the same is true for 25% of kidney cancers and over 80% of melanomas within temperate climatic regions, where solar ultraviolet effects are less dominant. For children, leukemias, cancers of the brain and spine, and osteosarcoma also correlate with radon exposure. If the observations are indicative of cause and effect then between 10 and 20% of incidence may be attributable to radon exposure at 50 Bq m^{-3}.

STATISTICAL UNCERTAINTY ANALYSIS OF RADON TRANSPORT IN NONISOTHERMAL, UNSATURATED SOILS* *(Wed., First Group, 10:40 a.m.)*

D. J. Holford, P. C. Owczarski, G. W. Gee, and H. D. Freeman

Pacific Northwest Laboratory, Richland, Washington

Transient radon flux from soil is affected by soil properties, as well as meteorological factors such as air pressure and temperature

*This work funded in part by the Department of Energy under Contract DE-AC06-76RLO 1830.

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changes at the soil surface and the infiltration of rainwater. Natural variations in meteorological factors and soil properties contribute to uncertainty in surface model predictions of radon flux. When integrated with a building transport model, these factors will add uncertainty to predictions of radon concentrations in homes.

A statistical uncertainty analysis using the Rn3D finite-element numerical model was conducted to assess the relative importance of these meteorological factors and the properties of the soil affecting the transport of radon. The Rn3D model has been enhanced to simulate the nonisothermal transport of radon by diffusion and advection in both the water and the air phases. Each input parameter was treated as a random variable, with either a uniform, normal, or lognormal distribution. Monte Carlo simulations were run after using Latin Hypercube Sampling to select random combinations of the model input parameters for each simulation. The relative contribution of each input parameter to the variability in radon flux predictions was assessed using multiple linear regression.

Identifying which input parameters contribute the most uncertainty to model predictions will indicate which parameters should be measured with the highest degree of spatial and temporal accuracy in the field.

"ORPHAN" RADON DAUGHTERS AT THE EPA SUPERFUND DENVER RADIUM SITE *(Wed., First Group, 2:25 p.m.)*

R. F. Holub, R. F. Droullard, and T. H. Davis

Bureau of Mines, Denver, Colorado

During 18 months of sampling airborne radioactivity at a National Priority List site in metropolitan Denver, the Bureau of Mines discovered radon daughters that are not supported by the parent radon gas. We refer to them as "orphan" daughters because the

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parent, radon, was not present in sufficient concentration to support the quantity of the measured daughter products. Measurements of the orphan daughters were made continuously, using the Bureau 3of Mines-developed radon and working level (radon daughter) monitors. The data showed high equilibrium factors, ranging from 0.7 to 3.5, for long periods of time. Additionally, repeated high-volume 15-minute grab samples were made using the modified Tsivoglou method. On average, 35% of the particulate activity was contributed by thoron daughters; the grab samples measured only radon daughters. Most of these samples had mixtures in which the ^{218}Po concentration was lower than that of ^{214}Pb and ^{214}Bi. This is in agreement with the high equilibrium factors obtained from the continuous sampling data. In view of the short half-life of radon progeny, we conclude that the source of the orphan daughters is in the vicinity of the Superfund site. The mechanism of this phenomenon is not understood at this time, but we will discuss its possible significance in the evaluation of population doses.

AIR CLEANING AND RADON DECAY PRODUCT MITIGATION *(Wed., First Group, 3:35 p.m.)*

P. K. Hopke, C.-S. Li, and M. Ramamurthi

Clarkson University, Potsdam, New York

We evaluated air cleaning as a means to mitigate risks arising from exposure to indoor radon progeny in several single-family houses in the northeastern United States, using a new, automated, semi-continuous activity-weighted size distribution measurement system. Measurements included radon concentration, condensation nuclei count, and activity-weighted size distribution of radon decay products. Measurements were made with and without the air cleaning system operating. The influence of particles generated by various sources common to normal indoor activities on radon progeny behavior was evaluated. Aerosols were generated by running water in a shower, burning candles, smoking cigarettes, vacuuming, opening doors, and cooking. Both a filtration unit and

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an electrostatic precipitator were evaluated. Using a room model, the changes in attachment rates, average attachment diameters, and deposition rates of the "unattached" fraction with and without the air cleaning systems were calculated.

DOSIMETRY OF RADON EXPOSURES: WHAT DOES IT IMPLY FOR RISK AND MEASUREMENT OF INDOOR EXPOSURE?* *(Invited Overview, Tues., 3:05 p.m.)*

A. C. James

Pacific Northwest Laboratory, Richland, Washington

Current estimates of lung-cancer risks resulting from the inhalation of radon and its progeny in homes are based on extrapolations of excess mortality observed in populations of underground miners. To project lung-cancer risk to the general public it is necessary to account for any effects that different exposure conditions may have on doses received by critical target cells in the respiratory tract. I will present the results of a review of aerosol, physiological and biological factors, and cells at risk, that are involved in evaluating critical doses from exposures in mine and indoor environments. Doses expressed per unit exposure to potential α-energy (in the common unit Working Level Month, WLM) are generally about 25% lower indoors than in underground mines for non-smoking healthy subjects. The variability of this dose conversion coefficient between different subjects (male and female adults, children and infants) is relatively slight. However, the variability of exposure conditions within and among homes, which is substantial, remains a dominant source of uncertainty in defining a "typical" comparative dose for domestic exposure.

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I will review the conversion from concentration of radon gas (the principal current metric of exposure in homes) to critical bronchial dose rate as well as the contribution to dose made by exposure to unattached radon progeny. Their implications for interpreting monitoring results will be discussed. Finally, the radon doses to other tissues and the dose per unit exposure to thoron progeny in mines and homes, and that from exposure to uranium ore dust, will be examined.

EFFECTS OF ACUTE RADON PROGENY EXPOSURE ON RAT ALVEOLAR MACROPHAGE NUMBER AND FUNCTION* *(Wed., Second Group, 11:20 a.m.)*

N. F. Johnson and G. J. Newton

Inhalation Toxicology Research Institute, Albuquerque, New Mexico

We exposed Fischer 344/N female rats to radon and radon progeny at a rate of 300 WLM/h for 3 h to determine whether alveolar macrophages (AM) are functionally impaired. Functional impairment of AM may be an ancillary event in the induction of pulmonary lesions and may also act as an indicator of dose to the peripheral lung. Alveolar macrophages were recovered by lavage from groups of exposed animals killed at 0, 2, 4, 8, 22, and 32 days after exposure; other groups of animals were exposed to air only. Cell numbers, viability, and cell differential counts were determined. The phagocytic activity of lavaged macrophages was determined using flow cytometry by measuring the uptake of fluorescein-labeled polystyrene latex microspheres (dia, 2 μm). Cell-cycle kinetics were also determined by flow cytometry using Hoechst 3342 fluorochrome. The number and viability of AM from exposed animals were similar to the values measured for control animals. There was no

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evidence of an inflammatory reaction during any period after radon-progeny exposure. Nuclear atypia, evidenced by increases in the numbers of binucleate cells and cells possessing micronuclei, occurred in AM 8 days after exposure; this response peaked at 22 days after exposure. Neither the phagocytic capability nor the cell-cycle kinetics of the AM were significantly affected at any time point after exposure. These results show that there is little impairment of AM function in rats after radon-progeny exposure; however, there may be interference with cell division, resulting in binucleate cells and cells containing micronuclei.

RADON--A HEALTH PROBLEM AND A COMMUNICATION PROBLEM *(Friday, 9:25 a.m.)*

R. H. Johnson

Key Technology, Inc., Jonestown, Pennsylvania

The Environmental Protection Agency (EPA) is making great efforts to alert the American public about the potential health risks of radon in homes. The news media have widely publicized radon as a problem; state and local governments are responding to public alarms; and hundreds of radon "experts" are now offering radon detection and remediation services. Apparently, EPA's communication program is working, and the public is becoming increasingly concerned with radon. But are they concerned with radon as a "health" problem in the way EPA intended? The answer is yes, partly. More and more, however, the concerns are about home resale values. Many homebuyers are now deciding whether to buy on the basis of a single radon screening measurement, comparing it with EPA's action guide of 4.0 pCi/L. They often conclude that 3.9 is OK, but 4.1 is not. Here is where the communication problems begin.

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The public largely misunderstands the significance of EPA's guidelines and the meaning of screening measurements. Seldom does anyone inquire about the quality of the measurements. Who asks about the uncertainty of reported radon measurements, or the results of EPA performance testing? Who asks about the uncertainty of lifetime exposure assessments based on a 1-hour, 1-day, 3-day, or even 30-day measurement? Who asks about the uncertainty of EPA's risk estimates?

Fortunately, an increasing number of radiation protection professionals are asking such questions. They find that EPA's risk projections are based on many assumptions which warrant further evaluation, particularly with regard to the combined risks of radon and cigarette-smoking. This is the next communication problem. What are these radiation protection professionals doing to understand the bases for radon health risk projections? Who is willing to communicate a balanced perspective to the public? Who is willing to communicate the uncertainty and conservatism in radon measurements and risk estimates? Can we call ourselves "radon experts" and in good conscience continue to ignore the growing public misunderstanding about radon? The general public needs our help to make appropriate decisions regarding their health and the value of their property. The call and the need are clearly evident for us to communicate what we know as professionals. Radon is becoming a "communication problem" and the challenge is for us to become communicators.

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CYTOTOXIC, CLASTOGENIC, AND MUTAGENIC RESPONSE OF MAMMALIAN CELLS EXPOSED IN VITRO TO RADON AND ITS PROGENY *(Wed., Second Group, 8:50 a.m.)*

R. F. Jostes,[1] E. W. Fleck,[2] R. A. Gies,[1] T. E. Hui,[1] T. L. Morgan,[1] J. L. Schwartz,[3] J. K. Wiencke,[4] and F. T. Cross[1]

[1]Pacific Northwest Laboratory, Richland, Washington; [2]Whitman College, Walla Walla, Washington; [3]Argonne National Laboratory, Argonne, Illinois; and [4]University of California, San Francisco, San Francisco, California

Preliminary data have been obtained using an *in vitro* radon exposure system developed at the Pacific Northwest Laboratory. We have compared our system with those of other laboratories for radon or simulated radon exposure, using a biological endpoint (cell survival) and a common cell line (CHO C-18). The survival response following exposure to radon and its progeny is an exponential function, with a D_0 of 61 cGy. Using a unified model for calculation of doses, this value is similar to that obtained at the University of Chicago using ^{212}Bi, and at Case Western Reserve University using radon and its progeny.

We have also investigated radon-induced mutations at the CHO-HGPRT locus. A linear dose response was observed, with induction of 1.4×10^{-6} mutations per viable cell per cGy. Mutants have been isolated after 76-cGy exposures and were evaluated using Southern blot techniques. These mutations are predominantly (48%) deletion events, with 24% showing no change from the parental line, and 28% showing a rearrangement of banding patterns. In contrast, spontaneous mutants exhibit a low percentage (11%) of deletion-type events, no alterations (0%), and 89% with no change.

We have investigated the time course of aberration induction after radon exposure of cycling human lymphocytes. We have noted before that low doses of radon induce a substantial mitotic delay (over 3 h with doses below 18 cGy). This is in contrast with the

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approximately 1-h delay per 100 cGy typically seen with low-LET x rays, and is in agreement with the longer delays reported after alpha-irradiation. In general, an aberration dose response occurred between 6.2 and 18.0 cGy, with an increase in the number of chromatid aberrations as metaphases are collected at later time intervals after irradiation. This may indicate that the more heavily damaged cells experience a longer delay.

INDOOR RADON SOURCES AND RADON MIGRATION IN THE SOIL *(Wed., First Group, 8:15 a.m.)*

G. Keller and M. Schütz

Universität des Saarlandes, Homburg/Saar, Federal Republic of Germany

High radon concentrations indoors are caused mainly by exhalation of radon from soil and ground water and its possible penetration into dwellings through breaks in the foundation. The "radon potential" of the soil is determined by its radium content, the radon emanating power, radon diffusion and other transport mechanisms. We have devised a special method of measuring these parameters and relating them to geological and meteorological conditions.

Inside a metal sphere, radon is allowed to decay to a short half-life, positively charged polonium ion, which is immediately deposited on a surface barrier detector. Alpha-spectroscopic analysis of its further decay allows us to precisely determine actual concentrations of ^{222}Rn and even of ^{220}Rn. The "geochemical radon potential" usually varies between 5 and 20 Bq L^{-1} of soil gas; the mean radon diffusion distance ranges from 0.5 m to 10 m. However, radon transported by water or gas flow through soil can migrate over tens of meters. Therefore, radon exhalation rates may be high even if the uranium content of the soil is low. In two soil areas with different uranium content, we found that radon exhalation rates varied between 2 and 2000 mBq m^{-2} s^{-1}.

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We have also measured indoor radon concentrations of about 8000 Bq m^{-3}, and as much as 36,000 Bq m^{-3} in a cellar. Based on the UNSCEAR dose-conversion factors, an indoor ^{222}Rn level of 8000 Bq m^{-3} yields an annual effective dose equivalent of about 200 mSv.

REANALYSIS OF DATA ON THE PARTICLE-SIZE DISTRIBUTION OF RADON PROGENY IN URANIUM MINES *(Tues., 2:20 p.m.)*

E. O. Knutson and A. C. George

Environmental Measurements Laboratory, New York, New York

We have reanalyzed 26 data sets from radon progeny particle-size measurements made in 1971 by George et al., in four New Mexico uranium mines. These data were obtained with a diffusion battery, which was new at the time and is still in use at our Laboratory.

The original data analysis, reported in 1975 with a supplement in 1977, was based on the *a priori* assumption of a unimodal log-normal size distribution. For new analysis we used a modern unfolding algorithm, in which this assumption is no longer needed. The new calculations show that 9 of the 26 distributions are, in fact, unimodal. (In these cases, the new calculations agree closely with the original.) Another 11 of the new distributions had a bimodal structure, with widely separated modes evoking the classical idea of attached and unattached radon progeny. The remaining cases showed bimodal structures, but the modes were not widely separated and were therefore not consistent with the classical picture.

To understand the implications of the recalculated distributions, we will combine the size distributions discussed above with an appropriate deposition equation which will yield dose conversion factors. These factors will be analyzed to see how they relate to the different locations and activities in the mines.

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AIR FILTRATION AND HEALTH RISK IN THE RESIDENCE *(Wed., First Group, 3:10 p.m.)*

B. C. Krafthefer and M. Schell

Honeywell, Inc., Golden Valley, Minnesota

When radon decays, the health risk is related to the concentration of both the attached and unattached fractions of the radon progeny. Mechanisms for removing these particles, such as filtration or deposition on surfaces in a room, will change the unattached fraction of progeny activity, and will therefore affect risk to the occupants in a complex manner.

We have completed a study on measuring the effects of particle concentration and the relative concentrations of attached and unattached decay products. We measured the impact of both active removal mechanisms, such as filtration, and passive mechanisms, such as deposition, on both fractions of the decay products. The results of using an electronic air cleaner for removal indicated overall reductions of the unattached fraction in a range of 10% to 20%, with a total working level reduction of around 70%.

The effect of using an electronic air cleaner on health risk was evaluated using lung deposition models, and the results were compared.

EXPERIMENTAL AND THEORETICAL STUDY OF THE FRACTION OF ^{210}PO ABSORBED IN GLASS

(Tues., 1:10 p.m.)

C. Landsheere, H. Vanmarcke, and A. Poffijn

Nuclear Physics Laboratory, Ghent, Belgium

In 1987, Lively suggested using measurement of the long-lived radon decay product ^{210}Pb ($T_{1/2}$ = 22.3 y) absorbed in glass surfaces to

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estimate long-term indoor radon concentrations. In 1988, Samuelsson developed a technique in which the alpha-decay of absorbed ^{210}Po in glass is used as an indicator of the radon exposure level during the last decades of exposure. This technique could be an important tool in epidemiological research in view of the long latency period between radon exposure and tumor manifestation.

The short-lived radon decay products ^{218}Po and ^{214}Po are both alpha-emitters. Due to the considerable energy of the recoiling atoms ^{214}Pb and ^{210}Pb (112 keV and 146 keV, respectively), 50% of the alpha-decays on surfaces such as vitreous glass leave progeny embedded in the surface beneath.

Lindhard theorizes how the range of heavy ions with low initial energy in an amorphous material can be determined. Using this theory, we calculated the distribution of ^{214}Pb and ^{210}Pb as a function of depth below the glass surface. We found that after alpha-decay of the absorbed ^{214}Po activity, 29.8% of the recoiling ^{210}Pb atoms escape from the glass.

By measuring the alpha-activity of the short-lived radon decay product ^{214}Po, we show that repeated cleaning of the glass removes the superficially deposited activity completely but allows the embedded activity to remain. Because the ^{210}Pb isotope is long-lived, a thin surface layer of the glass (approximately 0.055 μm thick) contains information about the integrated ^{222}Rn concentration.

In Samuelsson's retrospective exposure measurement technique, the alpha-activity of the ^{210}Po retained in glass is measured. However, the fraction of activity which is retained in glass depends on the values for the parameters of the room model. To examine the influence of the various parameters, we calculated the ratio of the ^{210}Po surface activity to the airborne ^{222}Rn activity after a period of 50 y. With the values of parameters that usually occur in houses, we found that this ratio ranges from 2% to 12%. It seems that the attachment rate constant is the dominant parameter: the surface

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activity is a factor of approximately three lower with an aerosol source (e.g., from smoking) in the room than without an aerosol source.

INVOLVEMENT OF GROWTH FACTORS AND THEIR RECEPTORS IN RADON-INDUCED RAT LUNG TUMORS *(Wed., Second Group, 10:55 a.m.)*

F. C. Leung, G. E. Dagle, and F. T. Cross

Pacific Northwest Laboratory, Richland, Washington

In this paper, we examine the role of growth factors (GF) and their receptors (GFR) in radon-induced rat lung tumors. Inhalation exposure of radon and its daughters induced lung tumors in rats, but the molecular/cellular mechanisms are not known. Recent evidence suggests that GF/GFR play a critical role in the growth and development of lung cancer in humans and animals. We have developed immunocytochemical methods for identifying sites of production and action of GF/GFR at the cellular level; for example, the avidin-biotin horseradish peroxidase technique. In radon-induced rat epidermoid carcinomas, epidermal growth factor (EGF), EGF-receptors (EGF-R), transforming growth factor-alpha (TGF-α), and bombesin were found to be abnormally expressed. These abnormal expressions, mainly associated with epidermoid carcinoma of the lung, were not found in any other lung tumor types. Our data suggest that EGF, EGF-R, TGF-α, and bombesin are involved in radon oncogenesis in rat lungs, especially in epidermoid carcinomas, possibly through the autocrine/paracrine pathway.

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MEASUREMENT OF SIZE DISTRIBUTIONS OF INDOOR RADIOACTIVE AEROSOLS *(Tues., 1:35 p.m.)*

C.-S. Li and P. K. Hopke

Clarkson University, Potsdam, New York

A major problem in evaluating the health risk posed by airborne radon progeny in indoor atmospheres is the lack of available information on the activity-weighted size distributions that occur in the domestic environment. With an automated, semi-continuous, graded screen array system, a series of measurements of activity-weighted size distributions have been made in several houses in the northeastern United States. Measurements were made in both an unoccupied house, in which human aerosol-generating activities were simulated, and in an occupied house, in which normal household activities were being conducted. The time evolution of the aerosol size distribution was measured in both situations. Results of these measurements and their implications for dose assessment will be presented.

PROTECTING THE PUBLIC AGAINST INDOOR EXPOSURE TO RADON *(Fri., 8:35 a.m.)*

F. Luykx,[1] A. Janssens,[1*] J. Sinnaeve,[2] and M. Olast[2]

[1]Commission of European Communities, Luxembourg; and [2]Commission of European Communities, Brussels, Belgium

On 21 February 1990, the Commission of European Communities (CEC) issued a "Recommendation on the Protection of the Public Against Indoor Exposure to Radon" because of increasing evidence and awareness of the problem. It is the Commission's task to

*Presenter

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coordinate provisions of Member States' control policies. We will discuss the different attitudes of these national authorities or advisory boards with respect to the reference level for remedial action in existing dwellings and the design level for future construction. The exact wording of the Recommendation is analyzed in order to understand the precise definition of such levels. We also emphasize the need for adequate public information and the application of optimization principles.

Because the levels are defined in terms of effective dose equivalents, we also address the considerations leading to the adopted reference value of 20 Bq m^{-3} mSv^{-1} y for conversion to radon concentrations.

For more than 10 y the Commission has supported and coordinated research on radon. We will also present information on three multinational projects and an epidemiological study related to radon exposure which are being carried out under the CEC's Radiation Protection Research Program for 1990-1991.

PREDICTION OF INDOOR RADON CONCENTRATION BASED ON RESIDENCE LOCATION AND CONSTRUCTION *(Tues., 9:55 a.m.)*

I. Mäkeläinen, A. Voutilainen, and O. Castrén

Finnish Centre for Radiation and Nuclear Safety, Helsinki, Finland

We have constructed a model for assessing indoor radon concentrations in houses where measurements cannot be performed. It has been used in an epidemiological study and to determine the radon potential of new building sites. The model is based on data from about 10,000 buildings. Integrated radon measurements were made during the cold season in all the houses; their geographic coordinates were also known. The 2-month measurement results were corrected to annual average concentrations. Construction data were collected from questionnaires completed by residents; geological data were determined from geological maps.

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Data were classified according to geographical, geological, and construction factors. In order to describe different radon production levels, the country was divided into three to seven regions. We assumed that the factors were multiplicative, and a linear concentration-prediction model was used.

The most significant factor in determining radon concentration was the geographical region, followed by soil type, year of construction, and type of foundation. The predicted indoor radon concentrations given by the model varied from 40 to 500 Bq m^{-3}. The lower figure represents a house with crawl space, built in the 1950s on clay soil, in the region with the lowest radon concentration levels. The higher value represents a house with a concrete slab in contact with the ground, built in the 1980s, on gravel, in the region with the highest average radon concentration.

AMERICAN LUNG ASSOCIATION'S RADON PUBLIC INFORMATION PROGRAM *(Fri., 9:00 a.m.)*

L. E. McCurdy

American Lung Association, Washington, DC

The American Lung Association (ALA), the nation's oldest voluntary health organization, is dedicated to the conquest of lung disease and the promotion of lung health. The objective of the ALA Radon Public Information Program is to reduce public exposure to elevated indoor radon levels through implementing grassroots-based radon public awareness campaigns by 22 local ALA groups. The program, which is funded by a grant from the U.S. Environmental Protection Agency (EPA), was initiated in December 1989; the first phase will continue until May, 1991. Activities of local chapters include distribution of free or reduced-cost radon kits; presenting programs in elementary and secondary schools; presenting information on TV news series and talk shows, and on radio Public Service Announcements and talk shows; presenting articles and feature stories in the print media; holding conferences, workshops, and displays at fairs

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and other exhibitions; distributing radon fact sheets through libraries and utility company mailings; and distributing videos through video chains and libraries. The local Lung Associations also serve as promoters for the EPA/Advertising Council Radon Public Service Announcement Campaign. We will highlight the activities of the groups in communicating radon health risks to the public; we will describe the results obtained and will attempt to evaluate the merits of the various approaches on the basis of the initial results.

PREVENTING EXCESSIVE RADON EXPOSURES IN U.K. HOUSING *(Wed., First Group, 4:50 p.m.)*

J. C. H. Miles, K. D. Cliff, B. M. R. Green, and D. W. Dixon

National Radiological Protection Board, Chilton, Didcot, Oxon, United Kingdom

In the United Kingdom (U.K.) it has been recognized for some years that some members of the population received excessive radiation exposures in their homes from radon and its decay products. To prevent such exposures, an Action Level of 400 Bq m^{-3} was adopted in 1987. In January, 1990, the National Radiological Protection Board (NRPB) advised that the Action Level should be reduced to 200 Bq m^{-3}, and this advice was accepted by the Government. It is estimated that exposures in up to 100,000 U.K. homes exceed this Action Level; this amounts to about 0.5% of the available housing. The U.K. authorities have developed a strategy for preventing such exposures: (1) Areas in which it is estimated that >1% of homes exceed the Action Level are being designated as Radon Affected Areas, and a program to map such areas is under way. Households in these areas are advised to have radon measurements made by NRPB under a "free" (Government-funded) scheme. (2) Householders found to have whole-house, whole-year average radon concentrations >200 Bq m^{-3} are advised to take remedial action, and are provided with information on how this can be done. Partial

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grants toward remedial work are available in cases of financial need. So far, around 3000 such households have been identified. (3) Within Affected Areas, localities are being defined where new homes must incorporate precautions against radon exposure.

In addition to this strategy, a joint case-control study of the risks of radon in homes is being undertaken by the Imperial Cancer Research Fund and NRPB, supported by the U.K. Government and the Commission of the European Communities.

MECHANISTIC MODELS OF CARCINOGENESIS: THEIR ROLE IN SCIENCE AND IN RISK ASSESSMENT *(Thurs., 8:15 a.m.)*

S. H. Moolgavkar

Fred Hutchinson Cancer Research Center, Seattle, Washington

The multistage model and the two-mutation clonal expansion model of carcinogenesis will be briefly reviewed. A linearized version of the multistage model is currently used by regulatory agencies for cancer risk assessment. A deficiency of the model is that it does not take explicit account of cell replication kinetics and is, therefore, unable to model the risk of non-genotoxic carcinogens. The two-mutation clonal expansion model, a generalization of the recessive oncogenesis model, has been shown to be consistent with a large body of epidemiologic and experimental data. It takes explicit account of cell replication kinetics. We will discuss mainly the latter model, with particular emphasis on its role in data analysis and in cancer risk assessment.

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